**References**

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RESPONSE: Most cases of false trigeminal neuralgia disappear within a few months after resection of causative lesions. Our hypothesis for this lengthy interim suggests that the arachnoid adhesion may be in the process of repairing such preoperative anatomical relationships as angulation of the cranial nerve axis or neurovascular compression. Interestingly, in cases presented by Haddad and Taha[1] and by Cappabianca, et al., trigeminal neuralgia disappeared after radical removal of the contralateral mass, but recurred after several months. We suggest that false trigeminal neuralgia caused by neurovascular compression mechanism may easily recur because the adhesive arachnoid membrane prevents the offending arterial loop from moving away from the affected cranial nerve roots, even after the displaced brainstem has returned to the normal position. Neurovascular compression theory for false trigeminal neuralgia is logical and can be applied to the cases reported by Haddad and Taha and by Cappabianca, et al. However, considering that no recurrent case has been reported besides these two, neurovascular compression seems to play a minor role in the development of contralateral trigeminal neuralgia.


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## Heat Shock Protein

TO THE EDITOR: We read with great interest the paper by Matz, et al. (Matz PG, Sundaresan S, Sharp FR, et al: Induction of HSP70 in rat brain following subarachnoid hemorrhage produced by endovascular perfusion. *J Neurosurg 85:*138–145, July, 1996). This is one of the first papers published on the relation of subarachnoid hemorrhage (SAH) and heat shock protein (HSP) induction in the brain.

Heat shock proteins and other stress proteins play an important role in stress responses of neurons, and it is generally agreed that HSPs have a neuroprotective function for the brain following injury, such as trauma, SAH, ischemia, and several degenerative diseases. It has been shown that successful induction of HSPs is critical to survival of neurons following ischemia, and its expression may also contribute to mechanisms of induced ischemic tolerance.[1] Heat shock proteins are expressed very soon after the injury, even before any changes are seen on light microscopy. Although the study by Matz, et al., was an experimental one, it is the beginning of an era in which stress proteins and other stress products will be clinically used as indicators for neuronal injury.

Although yet without clinical consequences, the induction of HSP after SAH is very interesting. If HSPs could be demonstrated in the cerebrospinal fluid (CSF) after SAH, then the time course and the amount of HSPs after SAH could be specified by simple examination of the CSF. Moreover, HSPs could serve as a marker for cerebral vasospasm not yet detected by transcranial Doppler. By defining the relation between the induction of HSPs and the clinical condition, high- and low- (surgical) risk patients could be determined.

We realize that the proposed ideas are just a theory and a great amount of work will be needed to explore the true clinical possibilities of HSPs as a valid marker and prognostic factor for patients with aneurysmal SAH. In the meantime, Matz, et al., are to be congratulated for their important and inspiring research.


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RESPONSE: We very much appreciate the comments of Drs. Kedaria and Menovsky concerning heat shock protein (HSP) expression in the cerebrospinal fluid (CSF). Although HSP induction and excretion into CSF has not been studied in detail, the hypothesis that Drs. Kedaria and Menovsky describe is an exciting one. Heat shock protein levels in CSF may subsequently prove to be a simple, accessible marker for following brain injury and recovery. Furthermore, the use of HSPs as an easily identifiable marker for cellular injury may ultimately aid in quantification of brain injury and prove to be a useful adjunct in the clinical decision process.


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